FAST-PRE (FACTORS THAT INCREASE RATE)

- Fever
- Anemia
- strenous exercise
- thyrotoxicosis
- pregnancy

FAAST-PRE (PPT FAILURE)

- Fever
- anemia
- Areythmia
- Strenous exercise
- Thyrotoxicosis
- Pregnancy

PRE-LOAD REDUCTION

- Sodium restriction
- Nitrates

AFTERLOAD REDUCTION

- – – Afterload reduction avoided in MS

GOLDEN RULE OF MANAGEMENT

1. Educate
2. constitute MDT
3. Treat the current problem
4. Slow down Progression and ongoing insult
5. Follow Up
6. Prevent, Identify and treat complications

DRUGS

- ACE has role in regurg murmurs
- BB has role in stenotic murmurs
- in regurg murmurs – mention failure drugs - ace, bb, diuretics

INTERVENTION

- – In stenotic lesions, Percutaneous intervention is preferred and if it is contraindicated, then Surgery is considered.
- – In Regurgitant lesions, Surgical intervention is preferred and if surgery is contraindicated, then percutaneous intervention is considered.
- regurg first preference is surgery
- stenotic - percutaneous options preferred

REPAIR VS REPLACEMENT

- Mitral lesions preferably repaired (in both situations - in MR, in MS)
- Aortic preferably replaced

MR INTERVENTION IS 60-40 RULE

AR INTERVENTION IS 50-50

- In MS patients, use the word of Pulmonary Congestion and in MR patients say signs of LV Dilatation.

4. Mechanical prosthetic valves

- Mitral mechanical prosthetics are more prone to thrombosis as compared to Aortic mechanical prosthetics, so INR is kept higher 2.5-3.5

NARROW PULSE PRESSURE

- means low cardiac output state

1. sign of severe aortic stenosis.
2. Severe mitral stenosis
3. hypovolaemlc state

SIGNS OF PULMONARY HTN

1. Raised JVP (with giant V waves due to tricuspid regurgitation)
2. Left parasternal heave and epigastric pulsation
3. Loud P2
4. Murmur of TR, Thrill may be present as well
5. Pul HTN –> Dilatation of Pulmonary artery –> PR –> EDM best heard in leaning forward (Graham Steel Murmur)
6. pulmonary congestion/cardiac failure (basal crepts)
7. Pedal Edema

- ACE has role in regurg murmurs
- BB has role in stenotic murmurs
- in regurg murmurs – mention failure drugs - ace, bb, diuretics
- regurg first preference is surgery
- Mitral lesions preferably repaired (in both situations - in MR, in MS)
- Aortic preferably replaced
- stenotic - percutaneous options preferred

MR INTERVENTION IS 60-40 RULE

AR INTERVENTION IS 50-50

INSPECTION

- (apart from subjective findings, focus mainly on 3 features)
- *PULSATION* - In 4 places precordium, Suprasternal notch and Right left supraclavicular area
- *SCARS* - Precordium, Axilla, legs, arms
- *DEFORMITY/ASYMMETRY/BULGE*
- inspection from Foot end
- inspection from right side, also ask patient to move arms aside and look in both axilla and also screen JVP here.
- DONT TOUCH THE PATIENT. WHENEVER HAVE TO TOUCH, INFORM THE PATIENT THAT I'M GOING TO TOUCH AT THIS PLACE ETC

PALPATION

- 4 major things
- *Apex beat* - location and character
- *Palpable heart sounds*
- *Right parasternal Heave*
- *Thrill*

AUSCULTATION

- Z route, don’t ask to hold breath, just ask to take a deep breath in or deep breath out

MURMUR DESCRIPTION

- Pitch
- Character
- Timing
- Area of maximum intensity
- Radiation
- Effect of respiration
- Effect of position
- Grade of murmur

RELEVANT

- stigmata of IEC, pulse, BP, JVP, Eyes, Chest, Pedal edema, Spleen, Ophthalmoscopy, postural drop
- pulse deficit, collapsing pulse, pistol shot femoral, quinke sign

---

- Description of Murmur should include

PITCH

- Only MS (?TS) is low pitch, all other murmurs are high pitched.

CHARACTER

- Soft, harsh, rumbling, blowing etc Most of stenotic murmurs are Harsh

TIMING

- Pansystolic - MR, TR, VSD
- Ejection Systolic - AS, PS (when severity increase, they may become pansystolic)
- Early diastolic - AR, PR (duration increase with severity)

- Mid Diastolic - MS, TS

AREA OF MAXIMUM INTENSITY

- Like all sounds, murmurs are audible in a range of area and we have to identify and mention the area where murmur intensity is maximum.
- This usually applies for systolic and Left sided murmurs.

GRADE

- For exam purpose murmurs are of grade 3 and 4 only. If thrill is present then describe it as grade 4 otherwise its grade 3

RADIATION

- Same concept as above.
- Usually minimal or no radiation of Diastolic and Right sided murmurs.

EFFECT OF RESPIRATION

- Right sided murmurs aggravated during inspiration.
- Left Sided murmurs aggravated during expiration.
- – – (No effect in VSD and PS)

EFFECT OF POSTURE WHERE APPLICABLE

- MS . Left lateral position, with bell. If exposure not allowed, Bell should be in contact with skin from underneath the shirt. A cloth between bell and skin will make it a diaphragm.
- AR . Ask patient to sit and lean over your hand.

INVESTIGATIONS

1. ECG – to look for (tell according to diagnosis)
2. CXR – to look for (tell according to diagnosis)
3. Echo – to confirm my diagnosis and establish etiology. To look for concomitant lesions and assess for hemodynamic consequences of (your diagnosis) and to device a treatment plan.

MANAGEMENT RULES

- I would like to assess functional ability of patient which begins with detailed history taking, investigations.
- I would educate the patient about nature and severity of illness, precautionary measures including dietary and lifestyle modification.
- Pharmacological treatment is directed towards controls of symptoms and prevention of complications. (Diuretics, Rate control, Anticoagulants, vasodilators etc)
- I would advise serial monitoring of illness and if it is progressing or symptoms are severe and persistent, I would advise surgical intervention.

MS

- – Diagnosis is Mitral stenosis complicated by pulmonary hypertension, functional tricuspid regurgitation and Right heart failure.
- – Patient is most likely in Sinus Rhythm.

DIFFERENTIALS?

- TS, Myxoma, Austin Flint)

CLINICAL FEATURES

1. MILD TO MODERATE

- Area 1 to 1.5
- asymptomatic or symptomatic on exertion

2. SEVERE

- area less than 1cm
- – exertional dyspnea, PND, orthopnea.
- – pulmonary htn, eventually RHF (JVP, Hepatomegaly, Ascites, Pedal edema)
- – atrial fibrillation
- – systemic embolisation (in upto 15%) – Renal failure, stroke, MI – mostly in pt in afib but sometimes also in sinus rhythm
- – compression by enlarged LA – Symptoms like hoarseness *ORTNER SYNDROME* (RLN), Cough (bronchus) , dysphagia (Esophagus) , hemoptysis
- – MS –> Pulmonary venous HTN –> Pulmonary congestion –> Pulmonary Arterial HTN

- – – – – some 20-30% patient may have depressed LV function due to rheumatic myocarditis that developed during rheumatic fever
- Syncope due to decreased output

SIGNS

- Malar rash
- JVP
- Apex
- parasternal heave
- S1
- P2
- Murmurs (MDM, Graham steel, PSM)
-

COMPLICATIONS

- AFib
- Systemic embolization
- Pulmonary HTN
- RHF
- Pul edema
- Complications of treatment (Drugs and valvotomy)

PROGNOSIS

1. Asymptomatic or Minimal

- 80% survival 10 yr

2. Limiting symptoms but untreated

- 15% survival 10 yr

3. Pulmonary hypertension

- mean survival upto 3 yr

CAUSES OF MS

- rheumatic heart disease.
- Calcification
- rarely mucopolysaccharidosis, carcinoid syndrome

- – MS in 99% cases is due to RHD. thickening of leaflet and fusion of commissure and calcium deposition
- – Calcium deposition in RHD is in leaflets, in dialysis patients calcium deposits in annulus and progress inwards to leaflets

IMPORTANCE OF INTENSITY OF S1

- Loud = Valve leaflets are pliable (opening snap is heard only when S1 is loud)
- Soft = Valve leaflets are calcified, it a sign of severity

SEVERITY OF MS

- Increase in Duration of murmur
- Signs of Pulmonary HTN
- signs of Pulmonary congestion
- Grahm Steel murmur of PR

HOW WILL YOU INVESTIGATE

- ECG – to look for AF, RVH, P-mitrale
- CXR – to look for straightening of left heart border, Double shadow in right heart, fullness of pulmonary conus with peripheral oligemia, Splaying of carina and left bronchus becoming horizontal, kerly B lines
- Echo – to confirm MS and establish etiology. To look for concomitant lesions and assess for hemodynamic consequences of Mitral Stenosis (valve area, Pulmonary HTN) and to calculate Wilkin scoring that will help me device a treatment plan.
- *– wilkin score is an imaging echo score measured in MS to assess pliability of mitral valve for feasibility of PTMC it includes 4 parameters . Leaflet thickening, calcification, mobility and Thickening of subvalvular apparatus. A score of less than 7 is good for PTMC, 8-11 is grey area and score of 12 or more is not good for PTMC*

HOW WILL YOU MANAGE

- Apart from clinical examination, I would like to assess functional ability of patient by taking a detailed history and obtaining investigations.
- I would educate the patient about nature and severity of illness, precautionary measures including dietary and lifestyle modification. restriction of activity, counseling about pregnancy.

PHARMACOTHERAPY

- Pharmacological treatment is directed towards controls of symptoms and prevention of complications.

1. Diuretics for congestion
2. Beta Blockers for rate control In AF
3. Anticoagulants if AF, LA Thrombus, history of Thromboembolic episode, Pul HTN
4. Vasodilators for Pul HTN

- – I would advise serial monitoring of illness and if it is progressing or symptoms are severe and persistent, when patient start getting Symptoms of nyha 2-3, I would advise intervention

5. Sodium restriction and Nitrates also have positive effect

INTERVENTION

1. Asymptomatic with moderate to severe MS and Pul Art pressure above 50 or wedge pressure above 25
2. Approach focuses on symptoms. like

- Episode of pulmonary edema
- Pulmonary HTN
- reduction in exercise capacity
- – Percutaneous mitral valvulopasty is first choice but MV Replacement is done if combined MS MR or Wilkins score above 12
- *– SOME CARDIOLOGISTS CONSIDER AFib TO BE AN INDICATION BUT NOT ALWAYS — ALSO, SYSTEMIC EMBOLIZATION IS NOT AN INDICATION IF SYMPTOMS ARE ONLY MILD OR MODERATE*

CRITERIA FOR PTMC

- Valve is mobile
- Valve calcification is mild
- No concomitant MR
- No thrombus in LA

INDICATIONS OF MVR

- – (in other words, these are contraindications of PTMC)
- valve heavily calcified
- MR
- LA thrombus

- concomitant coronary artery disease

TYPE OF VALVES

1. Mechanical

- – Mechanical mitral prosthetic valves are more prone to thrombosis than mechanical aortic prosthetic valves. The recommended INR range is thus higher (INR 2.5–3.5 or aver-age of 3.0). Low-dose aspirin should be used in conjunction with warfarin if the bleeding risk is not high

2. Bioprosthetic

- Warfarin be used for 6 months
- can't do PTMC if bioprosthetic valve gets stenosed. but can try percutaneous valve in valve procedure

AFIB

- Develops in 75% patients of MS
- 25% will have embolization if AFIB not treated
- Tachycardia further worsens output. so rate control is important.
- anticoagulation is also important. once AFib develops, warfarin should be given even if sinus rhythm is restored

MAZE PROCEDURE

- Done at time of Valvuloplasty.
- Endocardial incisions that disrupts electrical activity that sustains AFib

LA APPENDAGE

- Sewed to prevent a potential source of thrombus

MR

- – Diagnosis is Mitral regurgitation and Mitral Stenosis, complicated by pulmonary htn. Patient is not in heart failure and most likely patient is in Atrial Fibrillation.

- – – (Before presenting the case, say that I would like to complete my examination by counting pulse deficit and doing ophthalmoscopy, if not done in allotted time)

PATHOPHYSIOLOGY

- Mitral regurgitation results in a volume load on the heart (increases preload) and reduces afterload. The result is an enlarged LV with an increased EF. Over time, the stress of the volume overload reduces myocardial contractile function; when this occurs, there is a drop in EF and a rise in end systolic volume.

PREDOMINANT LESION

- Clinically, in mixed mitral valve disease, predominant lesion is to be decided by 3 parameters

1. Location of Apex Beat - undipslaced in predominant MS, Down and out in predominant MR
2. character of Apex Beat . Tapping in predominant MS, Thrusting in predominant MR
3. First Heart sound . Loud in Predominant MS, Soft in Predominant MR

- – (for example, if 2 parameters are in favour of MR, then predominant lesion is MR)

CAUSES OF MR

1. Acute

- MI
- IEC
- Complication during cardiac surgery

2. Chronic - Divided into primary and secondary

- – a) primary , When problem is in Valve and apparatus (leaflets, chordae tendineae, papillary muscles) mitral valve prolapse, IEC Rheumatic heard Disease, SLE, RA, Ankylosing spondylitis, Marfan Syndrome
- – b) Secondary - When LV dilates due to any reason, most commonly ischemia. This stretches valve annular - resulting in inadequate closure of valve.

DDS

- MR, TR, VSD

1. TR, murmur is Pansystolic but area of maximum intensity is at LLSB, it is aggravated during inspiration and doesn't radiate towards left axilla.
2. VSD, Murmur is Pansystolic but area of maximum intensity is Left sternal border and there is no effect of respiration.

INVESTIGATIONS

1. ECG to look for AF, LVH, Bifid P wave (LA enlargement) or Biphasic P wave (LA Hyoertrophy)
2. CXR to look for Cardiothoracic ratio, Fullness of pulmonary conus with peripheral oligemia, Double right heart border,
3. Echo – to confirm MR and establish etiology. To look for concomitant lesions and assess for hemodynamic consequences of MR (Pulmonary Hypertension, LV Dilatation, Ejection Fraction) and devise a treatment plan.

SEVERITY OF MR

- Heart sounds, Murmur, hemodynamic signs, peripheral signs)

1. Soft first heart sound, presence of S3 (sometimes S4, if rhythm is sinus)
2. Murmur - Thrill is sign of severity
3. Hemodynamic (Signs of Pulmonary hypertension and LV Dilatation)

- – Pulmonary Hypertension - Loud p2 (may be palpable as well), Left parasternal heave
- – LV Dilatation - Displaced Apex Beat, Presence of S3, chest crepts

4. Peripheral signs include high volume pulse (although its not a reliable sign in mitral valve lesions)

RUPTURE OF LEAFLET

- Anterior - murmur radiates to axilla
- posterior - murmur radiates to base and carotids

MANAGEMENT

- Apart from clinical examination, I would like to assess functional ability of patient by taking a detailed history and obtaining investigations.

- I would educate the patient about nature and severity of illness, precautionary measures including dietary and lifestyle modification. restriction of activity, counseling about pregnancy.

INDICATIONS OF INTERVENTION

- Lv dysfunction may develop before onset of symptoms, so LV dysfunction is also important criteria of intervention

1. Symptomatic MR is an indication for intervention.
2. or Asymptomatic patient with Ejection Fraction less than 60% and LVESD more than 40mm.
3. or patient has MR and undergoing open heart surgery for another cause like CABG or Aortic Valve replacement etc.
4. Asymptomatic patient with progressive deteriorating EF or increasing LVESD

TYPES OF INTERVENTION

1. Surgical

- Valve repair or valve replacement.
- – In MR, Repair is better because patient won't need lifelong anticoagulation, Risk of Infective Endocarditis is not high, subvalvular apparatus is preserved and LV geometry is not disturbed.
- – Repair or Replacement decision is usually made by surgeon at operating table after direct view of Valve and surrounding structures.

2. Transcutaneous intervention

- Can perform TEER (Transcutaneous End to End Repair) - Class 2a Indication

3. Mitra Clip

TREATMENT OF SECONDARY MR

- For secondary MR, treatment is GDMT (Guidelines Directed Management and Therapy)
- If GDMT response is inadequate, may consider TEER (class 2a indication).
- and if patient undergoing surgery for other reason, then also consider MV Surgery (Class 2b Indication)

POINTS

- – In Stenotic lesions, Auscultation findings are prominent but inspection findings are not prominent.
- – In Regurgitant lesions, inspection findings are also prominent.
- – In stenotic lesions, Percutaneous intervention is preferred and if it is contraindicated, then Surgery is considered.
- – In Regurgitant lesions, Surgical intervention is preferred in if surgery is contraindicated, then percutaneous intervention is considered.
- – In MS patients, use the word of Pulmonary Congestion and in MR patients say signs of LV Dilatation.

PULSE DEFICIT

- Counted when pulse is irregularly irregular.

1. Two person method

- One person count pulse and other person counts heart beat by auscultating at Apex. For 1 Minute.
- Difference between Apex Beat and Radial Pulse is Pulse Deficit.

2. One Person Method

- Auscultate at Apex and at same time palpate Radial pulse. Count the number of beats that are auscultated at Apex but not palpable in radial artery.
- – Deficit more than 10/minutes means A.Fib
- – Deficit less than 10/minutes means Multifocal Atrial Tachycardia or Premature contractions.

AS

- – Diagnosis is Aortic stenosis. Patient is not in heart failure and most likely patient is in sinus rhythm.

PATHOPHYSIOLOGY

1. Bicuspid

- manifest at lesser ages (50-60s)
- may have associated root dilatation
- offsprings will have 30% chance of having bicuspid AV

2. Calcification and degeneration

- Calcification process is similar to atherosclerosis.
- – risk factors include htn, hypercholesterolemia, smoking
- symptoms initially appear on exertion
- when area is 0.8cm, systole is prolonged. (reverse splitting of s2)
- eventually output reduced, LVH worsens.
- click is present when valve is still pliable.

DDS

- Pulmonary Stenosis
- HOCM
- Aortic Sclerosis

1. in PS, Apex is not heaving, P2 is soft and A2 is normal. maximum intensity of Ejection systolic murmur is in Pulmonic Area and radiation is to left neck.
2. in HOCM, double Apex Beat, Systolic thrill is in left lower para-sternal area and it doesn't radiates to neck.
3. in Aortic Sclerosis, Pulse is not low volume, Apex beat is not heaving and murmur doesn't radiate to neck.

GALLAVERDAN PHENOMENON

- Sometimes aorta is also calcified –> when high frequency component of AS murmur hits the Aortic wall it is reflected back and heard in Mitral area.
- – But its quality/character same and it is Ejection systolic murmur at mitral area, not Pansystolic.

CAUSES

- degenerative calcification
- Chronic Rheumatic heart disease
- Bicuspid Aortic Valve
- Congenital (Valvular, sub-valvular, supra-valvular)
- Infective Endocarditis

RISK FACTORS

- Hypertension, hypercholesterolemia, smoking

SEVERITY OF AS

1. Peripheral

- Low volume pulse
- slow rising pulse
- Narrow pulse pressure

2. Heart sounds

- soft or absent A2
- presence of S4
- reverse splitting of S2
- – – – CAUSES OF S2 reverse splitting (Severe AS, LBBB, HOCM, WPW type B)

3. Murmur

- Thrill
- prolonged duration and late peaking of murmur

4. Hemodynamic

- signs of Pulmonary hypertension
- signs of LV Failure

INVESTIGATIONS

1. ECG

- LV hypertrophy
- LV strain pattern
- Left atrial hypertrophy (bifid P waves in iead II)
- Left atrial dilatation (inverted of biphasic P waves inV1-V2)
- Left axis deviation
- Arrythmias or blocks

2. Chest X-ray

- LV enlargement
- Valve calcification
- post stenotic dilatation (or sometimes calcification) of ascending aorta
- Pulmonary congestion

3. Echocardiography

- to confirm AS and establish the etiology
- to assess hemodynamic consequences of AS (LVH and wall thickness) and also to devise a treatment plan.(doppler echo to assess gradient)

4. Cardiac catheterization

- to measure the gradient between left ventricle and aorta

5. Coronary Angiography to look for CAD.

COMPLICATIONS OF AS

1. Left ventricular failure
2. Pulmonary hypertension
3. Arrhythmias and Heart block
4. Infective endocarditis
5. Haemolytic anaemia
6. Heyde's syndrome (AS + angiodysplasia in Colon –> lower GI bleed –> iron deficiency anemia)
7. Sudden death

MANAGEMENT

- Apart from clinical examination, I would like to assess functional capacity of patient by taking a detailed history and obtaining investigations.
- I would educate the patient about nature and severity of illness, precautionary measures including dietary and lifestyle modification.

INDICATIONS OF SURGERY

1. Symptomatic
2. Asymptomatic but echo shows severe AS with LV Dysfunction
3. Asymptomatic but severe and undergoing CABG
4. (other - exercise BP drop, gradual EF decline, Nt-pro BNP)

- – – once intervention decided, then next decision is

1. When to intervene
2. what type of valve
3. What route

SAVR VS TAVI

- Age below 65 - SAVR is preferred
- Age above 80 - TAVI is preferred
- Age 65-80 - individualized decision

ROSS PROCEDURE IN BICUSPID

- Stenosed AV is replaced by pt own pulmonary valve, and PV is replaced by homograft or pt's bicuspid AV

BENTAL PROCEDURE

- If aorta root also dilated, then AV replacement wrapped in Dacron sheat and aorta replaced to valve

RISK AND WARFARIN TOLERABILITY

1. Risk not high

- Then assess tolerability and monitoring of warfarin
- – – a) warfarin acceptable - then we pick choice is Surgical AVR, type of prosthetic valve decided based upon age (mechanical if age less than 50, bioprosthetic if less than 65, if age 50-65, either can be used according to patient)
- – – b) warfarin not acceptable - then we do bioprothetic – option is to pick between SAVR and TAVI (TAVI for 80+ age, SAVR for age less than 65, between 65-80, either can be decided)

2. Surgical Risk high

- – – Life expectancy more than 1 year, anatomy favourable for TAVI, patient preference – Do TAVI
- – – Life expectancy less than 1 year, or anatomy not favourable for TAVI or patient refuses– Palliative care

TAVI PREREQUISITES

- Favourable valve and vessel anatomy
- Consideration of Life expectancy
- EF less rhan 25
- Not if active IEC
- not if concomitant AR
- not Bicuspid Valve

POINTS

- Typical symptoms are SAD (Syncope, Angina, Dyspnea/Death)
- Symptoms first start to appear after exertion.

CAUSE OF SYNCOPE IN AS?

- low cardiac output (Exercise,exertion causes peripheral vasodilation but LV cant increase output. This causes syncope. Same reason why vasodilators are contraindicated in AS)
- arrythmias

MECHANISM OF ANGINA IN AS

- left ventricular hypertrophy allows the left ventricle to maintain ejection fraction. LVH increases Oxygen demand.
- Coronary arteris flow depends upon duration of diastole. In AS, Systole is prolonged at the cost of diastole, compromising coronary flow.
- With LVH, LV End diastolic pressure is also increased.
- also, about half of patients also have CAD.

IMPORTANT

- Flow, Gradient and jet velocity determines severity

1. high Gradient

- 55+,velocity 5=super severr
- 40+, velocity 4 = severe

2. Low Gradient

- could be due to low flow or paradoxical.
- In Low gradient, provocative testing with dobitamine is done and 2 things are checked, Gradient and valve area.
- if gradient increase to 40+, without increase in valve area, it is called low Gradient low flow.
- if valve area increase, it means low flow is due to another cause like cardiomyopathy

ANTICOAGULATION

- bioprosthetic valve replaced surgically. 3-6 months anticoagulation
- percutaneously replaced.. 3-6 months dual antiplatelets

DAPT AFTER TAVI

- Clopidogrel 6 m + Low dose aspirin lifelong

AORTA ROOT DILATATION REPLACEMENT INDICATIONS

- If more than 5.5 cm
- if more than 5 and family h.o dissection or increasing 1cm in year
- if AVR to be done and root is 4.5+

AR

CAUSES

- RHD
- Bicuspid
- IEC
- HTN
- Aortic root dilatation (Marfan)
- Inflammatory (ankylosing spondylitis, RA, SLE etc)

PATHOPHYSIOLOGY

- The clinical course depend on the rapidity with which regurgitation develops. In chronic only a soft murmur
- With severity diastolic BP falls, and the LV progressively enlarges.
- Most patients remain asymptomatic for long periods even at this point.
- LV failure is a late event and may be sudden in onset. Exertional dyspnea, fatigue, paroxysmal nocturnal dyspnea and pulmonary edema may also occur
- Angina pectoris or atypical chest pain may occasionally be present.
- Associated CAD and presyncope or syncope are less com-mon than in aortic stenosis
- Hemodynamically, because of compensatory LV dilation, patients eject a large stroke volume, which is adequate to maintain forward cardiac output until late in the course of the disease
- LV diastolic pressure may rise when HF occurs. Abnormal LV systolic function as manifested by reduced EF (less than 50%) and increasing end-systolic LV volume (greater than 5.0 cm) are signs that surgical inter-vention is needed.

CLINICALLY

- Thrusting apex
- soft s2

- diastolic murmur at A2
- collapsing pulse
- wide PP
- Quinke
- Durozies
- Dancing carotid
- uvular pulsations
- Hill sign
- Austin Flint - Diastolic mumur mimicking MS

INTERVENTION

- *surgical repair is advised even in acute*
- symptoms
- any evidence of LV dysfunction (EF below 50 or LVESD above 50)
- undergoing other cardiac surgery
- – – options are same as for AS - SAVR vs TAVI

VSD

- On examination of the precordium. there ore no scars. There are no heaves, parasternal thrill
- The apex beat is undisplaced. On auscultation first and second heart sounds are normaal. There is a loud, harsh pansystolic murmur heard throughout the precordium and area of maximum intensity is at the lower left sternal edge.
- There is no peripheral stigmata of endocarditis. The pulse is 80 bpm, regular, normal volume and character. The blood pressure is 120/80mmHg.Venous pressure is not elevated and lung fields are dear to auscultation and there is no sacral or pedal oedema.
- – The diagnosis is a ventricular septal defect with a left-to-right shunt.

TYPES

1. According to comparison with aortic root

- mild restrtive - less than 25% of root diameter
- moderate restrictive - 25-75%
- UnRestrictive - more than 75%

2. according to left to right shunt

- less than 1.5 : 1
- 1.5-2.2 : 1

- more than 2.2 : 1

HEMODYNAMICALLY SIGNIFICANT

1. Pul HTN means VSD is Hemodynamically significant VSD , pay special attention to signs of Pulmonary HTN
2. LV volume overload –> displaced Apex beat, Thrusting character

POINT

- if there is also Pulmonary Stenosis or RVOT obstruction, this is good point as it delays development of Pul htn and eisenmenger

MANAGEMNET

- Small defects with *Flow in PA : Flow in AORTA less than 1.5 are managed conservatively and observed

INDICATIONS FOR VSD CLOSURE

- Increasing pulmonary:systemic blood flow ratio > 2:1
- LV dilatation or dysfunction
- Recurrent endocarditis
- Development of aortic regurgitation
- – Development of Eisenmenger is a contraindications of VSD Closure

PROCEDURES

- for closure of vsd, Patch is placed and sewed
- other option is to use devices for non surgical closure (specially for membranous VSD, rarely for VSD due to MI)

TOF

- On examination of precordium, there is no scar or visible pulsations. The apex beat is undisploced with a normal character. There is parasternal

heave and a systolic thrill at the upper left sternal edge. Pulmonic component of second heart sound is palpable. First and second heart sound are audible in their respective areas. There is a loud ejection systolic murmur at the upper left sternal edge aggravated in inspiration.

- There is a soft early diastolic murmur at the upper right sternal edge (aortic area) radiating down the sternal edge, which aggravated with patient sitting forward in expiration
- continuous murmur throughout systole· and diastole in the left subclavicular area and con also be heard posceriorly.
- There are no peripheral stigmata of endocarditis. There is central cyanosis and clubbing. The left radial pulse is diminished. The pulse rate is 80 bpm regular, normal volume and character.
- The left arm appears smaller than the right arm. The blood pressure is 120/80 in right arm and 100/75 in left arm.
- The venous pressure is not elevated
- The lung fields ore clear co auscultation and there is no pedal oedema.
- – The diagnosis is Fallot's tetralogy.

COMPONENT OF TOF

- VSD (with a right-to-left shunt)
- RVOT obstruction (Pulmonary Stenosis)
- RV hypertrophy
- Overriding aorta *(A DILATED AORTA THAT OVERRIDES THE SEPTUM)* - Sonetimes overrides 50% of septum resulting in double outlet for RV
- – – (if there is ASD also, it is called pentology of fallot)
- – – ASD + PS + RVH = Trilogy of Fallot

FEATURES

- Cyanotic spells (may lead to unconsciousness)
- Cyanosis, clubbing, Polycythemia
- Dyspnea
- growth retardation
- Seizures, CVA

PPT FACTORS OF CYANOTIC SPELLS

- Exercise
- Fever
- stress
- Hypoxia
- Dehydration

MANAGEMENT OF SPELL

- Knee position
- Oxygen
- Morphine
- Beta Blockers

INTERVENTION

1. Palliative

- Blood is not going into lungs, so blood from aorta is sent to lungs. this is achieved by connecting subclavian artery to pulmonary artery
- Classical - subclavian attached to PA
- Modified - A channel established between subclavian and PA
- – – *(results in low voluma radial pulse on that arm side)*

2. CORRECTION

- – (palliative channel is removed)
- Closure of VSD + patch correction for RVOT obstruction

POINTS

- – pulses if both arms should be compared. pulses are unequal if a procedure has been done in past (Sunclavian to PA anastamosis is done)
- – Murmur is of PS
- – No murmur due to VSD, because VSD is large –> RV and LV pressures are equal –> NO MURMUR
- – after correction, there is risk of serious arrhythmias caused by patches
- – – – also, in correction RVOT obstruction is corrected by placing a patch on Pul Valve that widens it – if this patch extends into Pul Artery - so there will be some PR - eventually there is risk of RV volume overload and RVH
- – – – in corrected patients, RV size assessment is needed every 1-2 year – if RV starts enlarging, it should be considered to be due to PR unless otherwise specified – an ealry PV replacement is done

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EISENMENGER

- Is a conditon with cyanosis and pulmonary htn due to intracardiac shunt

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ASD

- Fingers are not clubbed and there is no cyanosis.
- There are no upper limb developmental deformities.
- The pulse is 80bpm regular, normal volume and character.
- BP 120/75, Pulse pressure 45.
- Axillary Temp 99
- The venous pressure is not elevated.
- On examination of precordium, there ore no scars, deformity, bulge or flattening
- Apex is in left 5th ics in MCL.Second heart sound is not palpable. No parasternal heave.There is a systolic thrill at the upper left sternal edge.
- The first heart sound is normal and there is fixed wide splitting of the second heart sound.
- There is an ejection click and an ejection systolic murmur at the upper left sternal edge.
- The lung fields are dear to auscultation and there is no sacral or pedal oedema.
- The diagnosis is atrial septal defect with a left-to-right shunt.
- The absence of clinical signs of pulmonary hypertension would suggest this is a haemodynamically insignificant shunt

TYPES

- secundum 70% (fossa ovalis)
- primum 20% (defect also in atrioventricular septum, so there may b MR or TR also)
- venosus 10 (defect at site of SVC or IVC)
- Coronary (defect at coronory artery site)

PATHOPHYSIOLOGY

- initially L to R shunt – RV volume overload - RVH
- Eventually RV pressure increases - shunt reversal - Eisenmenger

CLINCAL

- Fixed wide split S2 (wide because RV volume overload so RV closure is delayed, Fixed because LA and RA pressures are equal)
- Loud P2
- In advance cases – left parasternal heave (RVH)

MURMURS

- – – 2 Murmurs , both are flow murmur - one is due to high flow from RA, second is due to high flow from RV
- MDM in Tricuspid - mimics MS but it is due to increased flow from RA through TV - *(this is high pitch murmur, MS murmur is low pitch)*
- ESM in pulmonary area – mimics PS but it is due to increased flow from RV through PV

FEATURES

- Dyspnea
- pain
- Pulmonary HTN and eisenmenger
- Failure
- Hemoptysis
- Areythmias
- Paradoxical emboli
- brain abscess

TREATMENT

- betablockers
- transatrial repair
- recent transcathetor repair
- – PFO closure is done if patient has hypoxemia on standing and no other cause found. otherwise PFO closure is not routinely done

INTERVENTION

- – if right side chambers are enlarged
- – if pulmoary to systemic flow ratio is more than 1.5 : 1

POINTS

- May cause eisenmenger
- paradoxical emboli
- ASD causes RBBB, secundum causes RBBB with RAD and Primum casues RBBB with LAD
- because right chambers enlarge, risk of AFib increased
- PFO closure or ASD closure with device increase the risk of AFib

IEC

- A gentleman lying in the bed at 45 angle. He has an iv branula on left hand and he was coperative during examination.
- He appears toxic. Fingers are clubbed and there are 2 splinter hemorrhages in left index finger.
- There is no cyanosis, pallor, jaundice, koilonychia, digital infarcts, lymphadenopathy or thyromegally.
- There are osler nodes on left hand and janeway lesions in right palm.
- Pulse rate is 108bpm, regular, low volume and there is no radioradial or radiofemoral delay.
- BP is 110/70
- RR is 28 and regular.
- Temp 100
- in pericardium, pulsations are visible just below left nipple. Apex beat is in left 5th ics in MCL. It is normal character. No thrill is palpable. First heart sound and second heart sound is normal and in Mitral area there is a PSM of grade 3 which is blowing. Best heard with diaphragm of stethoscope with patient in left lateral position holding his breath after expiration and murmur radiates towards axilla.
- Venous pressure is not elevated and chest is clear to auscultation.
- Abdomen is soft, non tender and there is no visceromegally.
- No sacral or pedal edema.
- My diagnosis is IEC.
- features in support of my diagnosis
- – – fever, tachycardia, tachypnea, clubbing, lymphadenopathy, janeway lesions, murmur of MR, splenomegaly

IEC

- bacterial or fungal infection. rarely aseptic
- more virulent organism produce acute febrile illness, valve regurgs and abscess formation

PREDISPOSING

- Congenital heard disease
- RHD
- – Regurg lesions are more prone to develop IEC than stenotic lesions

RISK FACTOR PROCEDURES

- high risk - Dental, respiratory, Urological, lower git
- low risk - Upper gi and pelvic procedures

DD

- tb, lymphoma, malaria

CAUSES

1. Native

- Aureus is most common cause
- streptococcal viridans
- fungal
- bartonella

2. Prosthetic

- first 2 months - staph (gram + and -ve)
-

ACUTE

- aureus, pseudomonas, candida, streptococcus, neisseria gonorrhea

CULTURE NEGATIVE

- prior axbx
- Actinebacterium, eikenella,
- fungal
- non infective (SLE, MARANTIC in CA lung)

CLINICAL FEATURES

- Few days to few weeks
- fever - typically high. may be low in old and ckd. and may b no fever.
- cough Dyspnea arthralgia myalgia
- tachycardia
- tachypnea
- *hands J-TOPICS*
- janeway lesions, Tender osler nodes, patechial hemorrhage, infarcts of digits, clubbing, splinter heamorhages,

IMMUNOLOGICAL

- Roth spots, osler nodes, hematuria, RA factor

VASCULAR

- janeway, digital infarcts, major arterial emboli, mycotic aneurysm, ICB

DUKES

- – Definite – 2 major, 1 major + 3 minor or 5 minor
- – Probable - 1 major + 1 minor, 3 minor
- – less likely - if illness settles in 4 days

MAJOR

- positive blood culture
- echo vegetations,new valvular lesion

MINOR

- predisposing heart condition or ivdu
- fever
- vascular phenomena
- immunological phenomena
- culture positive not meeting major
- echo positive not meeting major

WORKUP

- CBC
- CRP
- Blood culture . 3 samples from 3 different sites, 1 hour apart, taken after aseptic measure.. fungal cultures should be considered. 10% are culture negative
- ECHOCARDIOGRAPHY vegetations
- urine. hematuria proteinuria
- cxr . cardiomegaly or Kerley b lines
- ECG.. prolonged PR of root abscess
- RFT

TREATMENT

- empirical axbx before results of cultures
- ceftriaxone 2gm OD and vancomycin 1gm BD and ampicillin
- – – *(Native valve treatment for 4-6 weeks, prosthetic valve treatment is longer - for 6-8 weeks*

COMPLICATIONS (MEANS INDICATIONS OF SURGERY)

- Heart failure
- myocardial abscess
- refractory to axbx
- valvular obstruction
- repeated embolization
- low threshold if prosthetic valve IEC

ARF

- knee and ankle joints are swollen with warm and shiny skin. Joints are tender.
- There are subcutaneous nodules over bony prominences at elbow, spine.
- On leg, there is a pinkish rash, rounded with elevated margins consistent with erythema marginatum.

- on examination of precordium, there is no visible pulsation, scar mark or deformity. Apex beat is in left 5th ics in MCL and has normal character. No rub or thrill is palpable.
- First and second heart sounds are soft. In mitral area there is a PSM of grade 3. it is blowing and best heard with diaphragm of stethoscope and patient in left lateral position holding his breath after expiration. Murmur radiates towards axilla.
- fingers arw not clubbed, no central or peripheral cyanosis, no janeway lesions or osler nodes.
- Pulse
- Temp
- RR
- BP
- venous pressure is not elevated. chest is clear to auscultation. there is no cervical lymphadenopathy and no sacral or pedal edema.
- my diagnosis is Acute Rheumatic Fever.

CRITERIA

- 2 major , or 1 major + 2 minor
- – – Evidence of recent streptococcal infection
- ASOT > 200iu/mL
- history of scarlet fever
- positive throat swab
- increase in DNase B titre
- NAAT test
- Rapid antigen detection test - inexpensive and quick

MAJOR CRITERIA (MNEMONIC *CASES*)

- Carditis
- Poly Arthritis (Even Mono in high risk population)
- Subcutaneous nodules
- Erythema marginatum
- Sydenham's ch

MINOR CRITERIA

- Fever above 38.5 (38 in high risk population)
- ESR above 60 (30 in high risk population)
- PR Prolonged
- Arthralgia

INVESTIGATIONS

- CXR
- ECG
- Echo
- Aschoff bodies - inflammation with fibrinoid necrosis, lympho, plasma cells, enlarged macrophages with condensed nuclei termed Anitschkow cells

TREATMENT

- – GAS Eradication - Even if throat culture negative - Penicillin , Amoxicillin, - *Allergic to penicillin then give Cephalosporin, Azithro, Clinda*

SYMPTOMATIC

- Arthritis (Aspirin, NSAIDs or steroids)
- Carditis and Heart Failure.
- Erthema marginatum and Subcutaneous nodules are self limiting
- Chorea - Carbamezipine, valproate

SECONDARY PROPHYLAXIS

- im *penicillin G Benzathine* 28 days (21 days in high risk population)
- penicillin allergy - give macrolides or Sulfadiazine

1. No Carditis - 5 yrs or 21 age
2. Carditis + mild Valvulopathy - 10 yrs or 21 age
3. Moderate Valvulopathy - 10 yrs or 40 age

HEART FAILURE

LV

- Symptoms due to low output and pulmonary congestion due increase in pulmonary venous pressure
- Dyspnea is predominant symptoms
- – initially exertional dyspnea at first
- – – then progressing to orthopnea

- – – – paroxysmal nocturnal dyspnea
- – – – – Rest dyspnea
- Chronic nonproductive cough
- Nocturia due to excretion of fluid retained during the day and increased renal perfusion in the recumbent position
- fatigue
- exercise intolerance

RV

- symptoms due to volume overload
- Neck veins engorgement
- – – Sternal angle is 5cm above right atria. if JVP is 5cm above sternal angle, that means pressure is 10cm.
- – – upto 8cm is normal, above 8cm indicates fluid overload
- Ascites, Hepatomegaly
- Nausea and loss of appetite (gut congestion)
- Pedal Edema

CLINICAL SIGNS

- Dyspnea at minimal exertion
- cyanosis
- cachexia
- Sympathetic over activity (sweating and cold extremities)

CAUSES OF SYSTOLIC HEART FAILURE (EF BELOW 40)

1. IHD

- Ischemic cardiomyopathy

2. HTN

- an important cause
- also a ppt factor of HF that is due to other causes

3. Dilated cardiomyopathy

- MCC are alcohol and Myocarditis
- infiltrative

4. Valvular heart diseases

- specially Aortic valve

5. .

CAUSES OF DIASTOLIC HEART FAILURE (EF ABOVE 50)

1. HTN
2. Restrictive cardiomyopathy
3. Diabetes
4. pericardial diseases

STAGES

1. A

- patients at risk

2. B

- pt has Structural heart disease
- but no symptoms of failure

3. C and D

- pt with symptoms of failure

INVESTIGATIONS

1. CBC
2. RFTs
3. Electrolytes

- hypokalemia - increases risk of Arrythmias
- hyperkalemia - can’t use spironolactone
- hyponatremia - poor prognosis

4. Iron studies - HFE
5. Thyroid profile
6. BNP level

- less than 100 - failure unlikely
- more than 400 - indicates failure
- 100-400 - needs further evaluation

7. ECG
8. CXR
9. ECHO
10. CATH

POOR PROGNOSIS

1. Anemia
2. CKD
3. Hyponatremia
4. Cardiomegaly

TREATMENT

- Aimed at

1. Relieving Symptoms
2. Improving Functional ability
3. reducing hospitalization and mortality

HF WITH REF

- Diuretic + ACE
- early addition of BB and SGLT2

DIURETICS

- Mild overload - thiazide diuretics
- Moderate to severe - oral loop diuretics (in acute situation, give iv loop diuretics)
- potassium sparing (more beneficial if given in combination with thiazide or loop) - reduce mortality by 25%

RAAS INHIBITION

- improves mortality by 20%
- should also be used in HFrEF even if patient has no symptoms
- – – ACE inhibitors should be titrated to the dosages proved effective in clinical trials
- – – captopril 50 mg three times daily
- – – enalapril 10 mg twice daily
- – – ramipril 10 mg daily
- – – lisinopril 20 mg daily
- Asymptomatic hypotension is not indication of stopping ACE

SACUBITRIL AND VALSARTAN COMBINATION

- Reduce mortality by 20%
- – – Class I guidelines for the use of sacubitril/valsartan as a replacement for ACE inhibitors for patients with HF with reduced EF who remain symptomatic on an ACE inhibitor, beta-blocker, and mineralocorticoid inhibitor.
- – – Cause symptomatic hypotension
- – – should be started when SBP is above 100 and last dose of ACE was 36 hours ago

BETA BLOCKERS

- Carvedilol
- Extended release metoprolol (not short acting formulation)
- Bisoprolol
- – – started with low dose and dose gradually increased with 2 weeks intervals
- – – before starting, or before increasing the dose, Fluid retention must be evaluated.
- – – Should be avoided if patient has evidence of fluid retention
- – – daily weight monitoring

SGLT2

- Dapagliflozin and Empagliflozin
- additional benefit of improving proteinuria

DIGITALIS GLYCOSIDES

- Improve symptoms
- improve exercise tolerance
- reduce hospitalizations

NITRATE AND HYDRALAZINE

- combination produces better results in term of Hemodynamic control

IVABRADINE

- Stable heart failure

- patient already taking Ace, bb6, spironolactone and still symptomatic and rate above 75 – give Ivanradine

VERICIGUAT

- mortality benefit

ANTICOAGULATION

- DOACS and WARFARIN are equally effective

ANTI ARRYTHMIAS

- BB are sufficient
- rarel in some cases Amiodarone can be considered

STATINS

- No proven benefits wrt failure

POINTS

- COMBINATION THERAPY OF 4 DRUGS HAVING PROVEN MORTALITY BENEFIT

1. BB
2. SPIRONOLACTONE
3. SGLT2
4. SACUBITRIL-VALSARTAN

THERAPIES TO AVOID

- Thiazolidinediones
- CCBs
- combination of spironolactone, ACE, ARBs
- NSAIDs, cox

NON PHARMACOLOGICAL

ICD

- Specially if Arrythmias
- in HFrEF below 30-35%, even if no Arrythmias, improve mortality by 30%

RESYNCHRONIZATION

- – – Biventricular pacing
- Moderate to severe symptoms, EF below 35, Wide qrs more tha 120, LBBB pattern

DIET EXERCISE

- sodium upto 2.5gm
- exercise training

CORONARY REVASCULARIZATION

CARDIAC TRANSPLANT

- 1 year survival 80-90
- 5 year upto 75

OTHER SURGICAL

- Ventricle assist devices

PALLIATIVE

HF WITH PRESERVED EF

1. SGLT2 (Dapagliflozin)
2. Avoid fluid overload (Diuretics)

3. Control Comorbidities (HTN , DM, Arrythmias)

- – – ACE, ARBs, Sacubitril-Valsartan combination, Spironolactone, ICD, resynchronization – these therapies are not helpful in HFpEF

PPT FACTORS

INFECTION IN ISCHEMIA STOP FAST

- infections
- ischemia
- stopping the drugs
- fast rate
- Anemia
- Salt intake
- thyroid